Figure 2 shows the oligomerization of α-factor receptors during endocytosis. Fluorescence microscopy was used to localize GFP-tagged tailless receptors co-expressed with untagged wild type receptors (Δtail-GFP + WT) or alone (Δtail-GFP), GFP-tagged glucose transporters expressed with untagged wild type receptors (Hxt1-GFP + WT), and wild type receptors tagged with GFP (WT-GFP). Images were acquired before (0 min) or at the indicated times after the addition of agonist (α-factor, 5μM). Endosomal vesicles (E), the lysosome-like vacuole (V) and the endoplasmic reticulum (R) are indicated, as documented previously.

a1

Please replace the paragraph bridging pages 24 and 25 with the following paragraph:

FRET experiments were performed using truncated α-factor receptors lacking their cytoplasmic C-terminal regulatory domains (Ste2Δtail-CFP, donor; Ste2Δtail-YFP, acceptor), which are normal with respect to expression level, agonist binding affinity and G protein activation (8). Truncated receptors were used to reduce interfluorophore distance or mobility, improving the likelihood of detecting FRET, and to eliminate phosphorylation and ubiquitination of the receptor, which are required for desensitization and endocytosis. This ensured that FRET would not detect interactions between desensitized or internalized receptors.

a2

Please replace the first full paragraph on page 27 with the following paragraph:

If oligomerization is required for normal signaling, then cells expressing wild type receptors should signal inefficiently if they also overexpress signaling-defective receptors that interact with wild type receptors. Signal inhibition could occur if mutant receptor subunits interfere with the ability of wild type receptor subunits to undergo agonist induced conformational changes or to activate G protein heterotrimers. Accordingly, dominant-interfering mutant receptors were selected that inhibit signaling when overexpressed in cells expressing normal levels of wild type receptors. This approach identified a novel substitution (M250I) in transmembrane segment VI, a domain that controls activation of the α-factor receptor (9).

a3

On page 29, please replace the Reference list with the following list:

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a4